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Acenaphthene: Ambient Water Quality Criteria

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ACENAPHTHENE

Ambient Water Quality Criteria

Criteria and Standards Division
Office of Water Planning and Standards
U.S. Environmental Protection Agency
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Section 304(a) of the Clean Water Act (33 U.S.C. 1314(a)), requires EPA to publish and periodically update water quality criteria. These criteria are to reflect the latest scientific knowledge on the identifiable effects of pollutants on public health and welfare, aquatic life, and recreation. This report presents water quality criteria for acenaphthene. It presents concentration criteria for the protection of fresh water and saltwater aquatic life. It presents 'safe' concentrations for humans, and in the case of suspect or proven carcinogens, gives various levels of incremental cancer risk. A section 304(a) water quality criterion is a qualitative or quantitative estimate of the concentration of a water constituent or pollutant in ambient waters which, when not exceeded, will ensure a water quality sufficient to protect a specified water use. Under the Act a criterion is a scientific entity, based solely on data and scientific judgement. It does not reflect consi....

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CRITERION DOCUMENT

ACENAPHTHENE

CRITERIA

Aquatic Life

The data base for freshwater aquatic life is insufficient to allow use of the Guidelines. The following recommendation is inferred from toxicity data for saltwater organisms.

For acenaphthene the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 110 µg/l as a 24-hour average and the concentration should not exceed 240 µg/l at any time.

For acenaphthene the criterion to protect saltwater aquatic life as derived using the Guidelines is 7.5 µg/l as a 24-hour average and the concentration should not exceed 17 µg/l at any time.

Human Health

For the prevention of adverse effects due to the organoleptic properties of acenaphthene in water, the criterion recommended is .02 mg/l.

Introduction

Acenaphthene (1,2-dehydro-acenaphthylene or 1,8-ethylene-naphthalene) occurs in coal tar produced during the high temperature carbonization or coking of coal. It is used as a dye intermediate in the manufacture of some plastics, as an insecticide and fungicide, and has been detected in cigarette smoke and gasoline exhaust condensates. Acenaphthene is polynuclear aromatic hydrocarbon with a molecular weight of 154 and a formula of $C_{12}H_{10}$.

The compound is a white crystalline solid at room temperature with a melting range of 95 to 97°C and a boiling range of 278 to 280°C (Lidner, 1931). The vapor pressure is less than 0.02 mm Hg. Acenaphthene is soluble in water (100 mg/l), but solubility increases in organic solvents such as ethanol, toluene, and chloroform.

Acenaphthene will react with molecular oxygen in the presence of alkali-earth metal bromides to form acenaphthequinone (Digurov, et al. 1970). In the presence of alkali-earth metal hydroxides, acenaphthene reacts with ozone to produce 1,8-naphthaldehyde carboxylic acid (Menyailo, et al. 1971). Acenaphthalene can be oxidized to aromatic alcohols and ketones using transition metal compounds as catalysts (Yakobi, 1974). Acenaphthene is stable under laboratory conditions and resists photochemical degradation in soil stability studies (Medvedev and Davydow, 1972).

Acenaphthene has been demonstrated to affect the growth of plants through improper nuclear division and polypoidal chromosome number. These same observations were noted in

several microorganisms as well. Little information regarding aquatic toxicity was found. The freshwater acute value for bluegill was 1,700 µg/l, and the bioconcentration factor was 397. Saltwater toxicity to the sheepshead minnow was 2,230 µg/l, and no bioconcentration data were available. Data on toxicity to non-human mammals were few and virtually no incidences of human acenaphthene toxicity were noted. There was some information found showing organoleptic effects attributed to acenaphthene in water. A detection range of 0.02 to 0.22 mg/l was given. Laboratory experimentation points out the possibility of limited metabolism of acenaphthene to napthalic acid and napatholic anhydride.

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AQUATIC LIFE TOXICOLOGY*

FRESHWATER ORGANISMS

Introduction

The data base for acenaphthene and freshwater organisms is limited to a few acute toxicity tests under static conditions with unmeasured concentrations and no criterion can be derived from these results. A bioconcentration test has been conducted for 28 days and the depuration rate was determined.

Acute Toxicity

The bluegill has been exposed to acutely lethal concentrations of acenaphthene (U.S. EPA, 1978) and the resulting adjusted 96-hour LC50 value is 929 µg/l (Table 1). After use of the sensitivity factor (3.9), this result leads to the Final Fish Acute Value of 240 µg/l.

An acute test with Daphnia magna resulted in an adjusted 48-hour EC50 of 34,900 µg/l (Table 2) and the Final Invertebrate Acute Value derived from that datum is 1,700 µg/l. Since the

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*The reader is referred to the Guidelines for Deriving Water Quality Criteria for the Protection of Aquatic Life [43 FR 21506 (May 18, 1978) and 43 FR 29028 (July 5, 1978)] in order to better understand the following discussion and recommendation. The following tables contain the appropriate data that were found in the literature, and at the bottom of each table are the calculations for deriving various measures of toxicity as described in the Guidelines.

equivalent value (240 µg/l) for fish is lower, it becomes the Final Acute Value.

Chronic Toxicity

No chronic tests have been reported for acenaphthene and freshwater organisms.

Plant Effects

The alga, Selenastrum capricornutum, appears to be rather sensitive. The 96-hour EC50 values for chlorophyll a and cell numbers are 530 and 520 µg/l, respectively (Table 3). The Final Plant Value is 520 µg/l.

Residues

The bluegill accumulated acenaphthene during a 28-day exposure (U.S. EPA, 1978). The bioconcentration factor was 387 using ^{14}C-acenaphthene and thin-layer chromatography for verification (Table 4). The half-life of this chemical in the whole body was less than 1 day.

CRITERION FORMULATION

Freshwater Aquatic Life

Summary of Available Data

The concentrations below have been rounded to two significant figures.

Final Fish Acute Value = 240 µg/l

Final Invertebrate Acute Value = 1,700 µg/l

Final Acute Value = 240 µg/l

Final Fish Chronic Value = not available

Final Invertebrate Chronic Value = not available

Final Plant Value = 520 µg/l

Residue Limited Toxicant Concentration = not available

Final Chronic Value = 520 µg/l

0.44 x Final Acute Value = 110 µg/l

No freshwater criterion can be derived for acenaphthene using the Guidelines because no Final Chronic Value for either fish or invertebrate species or a good substitute for either value is available.

Results obtained with acenaphthene and saltwater organisms indicate how a criterion may be derived.

For acenaphthene and saltwater organisms, 0.44 times the Final Acute Value is less than the Final Chronic Value which is derived from results of an embryo-larval test with the sheepshead minnow. Therefore, it seems reasonable to estimate a criterion for acenaphthene and freshwater organisms using 0.44 times the Final Acute Value.

The maximum concentration of acenaphthene is the Final Acute Value of 240 µg/l and the 24-hour average concentration is 0.44

times the Final Acute Value. No important adverse effects on freshwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

CRITERION: For acenaphthene the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 110 µg/l as a 24-hour average and the concentration should not exceed 240 µg/l at any time.

Table 1. Freshwater fish acute values for acenaphthene (U.S. EPA, 1978)

Organism	Bioassay Method*	Test Conc.**	Time (hrs)	LC50 (ug/l)	Adjusted LC50 (ug/l)
Bluegill, *Lepomis macrochirus*	S	U	96	1,700	929

* S = static

** U = unmeasured

Geometric mean of adjusted values = 929 µg/l $\frac{929}{3.9}$ = 240 µg/l

Table 2. Freshwater invertebrate acute values for acenaphthene (U.S. EPA, 1978)

Organism	Bioassay Method*	Test Conc.**	Time (hrs)	LC50 (ug/l)	Adjusted LC50 (ug/l)
Cladoceran, Daphnia magna	S	U	48	41,200	34,900

* S = static

** U = unmeasured

Geometric mean of adjusted values = 34,900 µg/l $\frac{34,900}{21}$ = 1,700 µg/l

Table 3. Freshwater plant effects for acenaphthene (U.S. EPA, 1978)

Organism	Effect	Concentration (ug/l)
Alga, Selenastrum capricornutum	96-hr EC50 chlorophyll a	530
Alga, Selenastrum capricornutum	96-hr EC50 cell numbers	520

Lowest plant value = 520 µg/l

Table 4. Freshwater residues for acenaphthene (U.S. EPA, 1978)

Organism	Bioconcentration Factor	Time (days)
Bluegill, *Lepomis macrochirus*	387	28

SALTWATER ORGANUAA)

Introduction

As with freshwater organisms there is a limited data base for acenaphthene and saltwater organisms. There is little difference between LC50 and EC50 values for the sheepshead minnow, Cyprinodon variegatus, the mysid shrimp, Mysidopsis bahia, or the alga, Selenastrum costatum. An embryo-larval test has been conducted with the sheepshead minnow.

Acute Toxicity

The adjusted 96-hour LC50 value for the sheepshead minnow is 1,219 µg/l (Table 5). Based on this datum, the Final Fish Acute Value for acenaphthene and saltwater fish is 330 µg/l.

For the mysid shrimp (U.S. EPA, 1978) the 96-hour LC50 is 821 µg/l (Table 6). The Final Invertebrate Acute Value is 17 µg/l, and, since this concentration is lower than the comparable value for fish, it also becomes the Final Acute Value.

Chronic Toxicity

The ratio of acute and embryo-larval test results with the sheepshead minnow is small. The unadjusted 96-hour LC50 was 2,230 µg/l (Table 5) and the geometric mean of the no-effect and effect concentrations was 710 µg/l (Table 7). The chronic value, derived by dividing this geometric mean by 2, is 355 µg/l. When this concentration is divided by the sensitivity factor (6.7), the Final Fish Chronic Value of 53 µg/l is obtained.

Plant Effects

As discussed earlier, the alga, Skeletonema costatum, is as sensitive as the sheepshead minnow and the mysid shrimp. The 96-hour EC50 value for chlorophyll a and cell numbers is 500 µg/l. This also is the Final Plant Value.

CRITERION FORMULATION

Saltwater Aquatic Life

Summary of Available Data

The concentrations below have been rounded to two significant figures.

Final Fish Acute Value = 330 µg/l

Final Invertebrate Acute Value = 17 µg/l

Final Acute Value = 17 µg/l

Final Fish Chronic Value = 53 µg/l

Final Invertebrate Chronic Value = not available

Final Plant Value = 500 µg/l

Residue Limited Toxicant Concentration = not available

Final Chronic Value = 53 µg/l

0.44 x Final Acute Value = 7.5 µg/l

The maximum concentration of acenaphthene is the Final Acute Value of 17 µg/l and the 24-hour average concentration is 0.44 times the Final Acute Value. No important adverse effects on saltwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

CRITERION: For acenaphthene the criterion to protect saltwater aquatic life as derived using the Guidelines is 7.5 µg/l as a 24-hour average and the concentration should not exceed 17 µg/l at any time.

Table 5. Marine fish acute values for acenaphthene (U.S. EPA, 1978)

Organism	Bioassay Method*	Test Conc.**	Time (hrs)	LC50 (ug/l)	Adjusted LC50 (ug/l)
Sheepshead minnow, Cyprinodon variegatus	S	U	96	2,230	1,219

* S = static

** U = unmeasured

Geometric mean of adjusted values = 1,219 µg/l $\frac{1,219}{3.7}$ = 330 µg/l

Table 6. Marine invertebrate acute values for acenaphthene (U.S. EPA, 1978)

Organism	Bioassay Method*	Test Conc.**	Time (hrs)	LC50 (ug/l)	Adjusted LC50 (ug/l)
Mysid shrimp, *Mysidopsis bahia*	S	U	96	970	821

* S = static

** U = unmeasured

Geometric mean of adjusted values = 821 µg/l $\frac{821}{49}$ = 17 µg/l

Table 7. Marine fish chronic values for acenaphthene (U.S. EPA, 1978)

Organism	Test*	Limits (ug/l)	Chronic Value (ug/l)
Sheepshead minnow, *Cyprinodon variegatus*	E-L	520-970	355

* E-L = embryo-larval

Geometric mean of chronic values = 355 µg/l $\quad \frac{355}{6.7} = 53$ µg/l

Lowest chronic value = 355 µg/l

TABLE 1 (cont.)

ORGANISM	TREATMENT	EFFECTS NOTED	REFERENCE
Bacteria:			
Mycobacterium rubrum	Vapors from 10-20 mg crystals	Elongation and thickening of cells; unstable polyploidy	Imshenetsky and Zhil'tsova, 1973
Rhizobium	Vapors	Increase in DNA content; change in biochemical properties	Avvakumova, et al. 1975
Algae:			
Chara globularis; *Nitella flagelliformis*	Saturated solution (12-120 hours)	Number of cells in mitosis reduced; chromosomes clumped at metaphase; chromosomes doubled	Sarma and Tripathi, 1976a, b

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Mammalian Toxicology and Human Health Effects

EXPOSURE

Ingestion from Water

Acenaphthene has been detected in the effluent from petrochemical, pesticide, and wood preservative industries by U.S. Environmental Protection Agency monitoring studies (U.S. EPA, 1978b). A survey of organic chemical monitoring data from a variety of published and unpublished sources indicated that acenaphthene had been identified in 11 studies (U.S. EPA, 1976). Seven of these studies analyzed effluent from petrochemical or wood preserving plants, while two identified the chemical in finished drinking water, and another study found it in a river sample. An analysis of the settling pond water from a wood preserving plant showed acenaphthene present at a level of 0.2 mg/l (U.S. EPA, 1973). Acenaphthene was also identified by two Russian authors as one of several organic compounds in the wastewater from by-product coke manufacture (Andreikova and Kogan, 1977).

In an examination of water extracted by macroreticular resins from a contaminated well in Ames, Iowa, investigators isolated acenaphthene at a level of 1.7 ppm (Burnham, et al. 1972). Identification was verified by comparison with mass spectrum, retention time, and ultraviolet spectrum of a standard. The authors (Burnham, et al. 1972) noted that the contamination is believed to be the result of residue from a coal gas plant which may have leached into the aquifer after the plant closed in 1930. Meijers and Van der Leer (1976) detected acenaphthene by gas chromatography in a

20-liter sample of water from the river Maas in the Netherlands. Although not quantified by the authors, acenaphthene was a minor constituent of the PAH mixture identified in the water. Acenaphthene has a low solubility in water, but its presence in water may be significant due to possible adsorption on particulates.

Ingestion from Foods

Only one study (Onuska, et al. 1976) was found on the occurrence of acenaphthene in foods. Levels of $\geq$3.2 µg acenaphthene/kg (the detection limit) were presumptively identified in the tissues of shellfish of an unspecified species and location. Relative to other PAHs detected in this sample, the amount of acenaphthene was small.

A bioconcentration factor (BCF) relates the concentration of a chemical in water to the concentration in aquatic organisms, but BCF's are not available for the edible portions of all four major groups of aquatic organisms consumed in the United States. Since data indicate that the BCF for lipid-soluble compounds is proportional to percent lipids, BCF's can be adjusted to edible portions using data on percent lipids and the amounts of various species consumed by Americans. A recent survey on fish and shellfish consumption in the United States (Cordle, et al. 1978) found that the per capita consumption is 18.7 g/day. From the data on the nineteen major species identified in the survey and data on the fat content of the edible portion of these species (Sidwell, et al. 1974), the relative consumption of the four major groups and the weighted average percent lipids for each

group can be calculated:

Group	Consumption (Percent)	Weighted Average Percent Lipids
Freshwater fishes	12	4.8
Saltwater fishes	61	2.3
Saltwater molluscs	9	1.2
Saltwater decapods	18	1.2

Using the percentages for consumption and lipids for each of these groups, the weighted average percent lipids is 2.3 for consumed fish and shellfish.

A measured steady-state bioconcentration factor of 387 was obtained for acenaphthene using bluegills containing about one percent lipids (U.S. EPA, 1978a). An adjustment factor of 2.3/1.0 = 2.3 can be used to adjust the measured BCF from the 1.0 percent lipids of the bluegill to the 2.3 percent lipids that is the weighted average for consumed fish and shellfish. Thus, the weighted average bioconcentration factor for acenaphthene and the edible portion of all aquatic organisms consumed by Americans is calculated to be 387 x 2.3 = 890.

Inhalation

Acenaphthene has been identified as one of many polycyclic aromatic hydrocarbons (PAHs) in gasoline exhaust condensate (Grimmer, et al. 1977) and cigarette smoke condensate (Harke, et al. 1976; Severson, et al. 1976). However, no estimates have been made of the degree of exposure to acenaphthene that occurs to individuals inhaling cigarette smoke or gasoline exhaust.

A 420,000 cubic foot sample of air in Sydney, Australia, was found to contain 3.9 ppm of solid acenaphthene, or 0.07 µg/100 m^3 (Cleary, 1962), indicating that individuals in urban environments may be exposed to measurable levels of acenaphthene.

Dermal

No information is available on dermal exposure to acenaphthene.

PHARMACOKINETICS

Absorption

No data are available on the absorption of acenaphthene.

Distribution

No data are available on the distribution of acenaphthene.

Metabolism

Chang and Young (1943) isolated, by several methods, the anhydride of naphthalene-1,8-dicarboxylic acid from the urine of two groups of male white rats administered acenaphthene orally. One group of rats was fed twice a day on a stock diet containing 1 percent acenaphthene; a second group was dosed by gavage on alternate days with 1 ml of a fine suspension of 0.1 g acenaphthene in dilute starch solution. The authors raised the possibility that the naphthalic anhydride is a decomposition product of conjugated compounds arising from the acid used in the extraction procedure, rather than a metabolic product of acenaphthene. No acenaphthene was detected in the urine of the rats.

Aside from this study, no other data were found concerning the metabolism of acenaphthene.

Excretion

As indicated previously, no acenaphthene was found in the acidified urine of rats dosed orally with acenaphthene (Chang and Young, 1943). No other data are available on the excretion of acenaphthene.

EFFECTS

Acute, Subacute, and Chronic Toxicity

Very little is known about the human toxicity of acenaphthene. It is irritating to skin and mucous membranes, and may cause vomiting if swallowed in large quantities (Sax, 1975).

Similarly, limited data are available on the toxic effects of acenaphthene in mammals. Knobloch, et al. (1969) investigated the acute and subacute toxic effects of acenaphthene in rats and mice. Two g acenaphthene per kg body weight administered orally in olive oil to seven young rats (sex not specified) on a daily basis for 32 days caused loss of body weight, changes in peripheral blood, heightened aminotransferase levels in blood serum, and mild morphological damage to both the liver and kidney. An LD_{50} of 10 g/kg was reported for rats and 2.1 g/kg for mice. The authors (Knobloch, et al. 1969) noted that the morphological damage to the kidney and liver was greater when acenaphthene was administered in a subacute manner than when an acute dose was given. After 32 days the animals showed mild bronchitis and localized inflammation of the peribronchial tissue.

In another toxicity study, Reshetyuk, et al. (1970) exposed 100 rats to a five-month chronic inhalation of acenaphthene at a level of 12 ± 1.5 mg/m^3 for four hours a day, six days per week. Toxic effects to the blood, lungs, and glandular constituents were reported. The bronchial epithelium showed hyperplasia and metaplasia, possibly a symptom of the pneumonia which killed a large number of animals. However, no signs of malignancy appeared during the 13 months of exposure. Reshetyuk, et al. (1970) also reported an LD_{50} of 600 ± 60 mg/kg for rats given intraperitoneal injections of acenaphthene. It must be pointed out, however, that the lack of reported controls, as well as the inadequate and confusing description of methods, make this study unsuitable as the basis for a criterion.

Gershbein (1975) investigated the effect of acenaphthene and many other hydrocarbons upon the degree of liver regeneration in partially hepatectomized male rats. Acenaphthene in peanut oil was injected subcutaneously into one group of animals on a daily basis for seven days following surgery for total dose of 5 to 20 mM/kg. A second group of animals was administered the chemical as part of the diet at 0.03 and 0.10 percent (by weight). Ten days following the surgical treatment, all animals were sacrificed and the liver weights determined. Liver regeneration was significantly ($p < 0.01$) accelerated in both the injection-treated animals and the higher oral dose group. A third group of rats were injected with acenaphthene three times and then sacrificed 72 hours after surgery. Among all those exposed in this manner to

5 polycyclic hydrocarbons, acenaphthene-treated animals were the only animals showing a significant acceleration of liver regeneration. These results are in contrast to an earlier study by Gershbein (1958), in which a low dose of 4.6 mM acenaphthene per kg did not result in a significant liver regeneration acceleration. In the 1958 study, only a dose of 31.8 mM/kg induced a significant regeneration.

Although the toxic effects of acenaphthene are not well documented, the reactions of humans to an odor from an aqueous solution of the chemical, which may result in rejection of the contaminated water, have been investigated. In a study of the odor thresholds of organic pollutants (Lillard and Powers, 1975), a panel of 14 judges detected acenaphthene at a mean threshold of .08 ppm, with a range of 0.02 to 0.22 ppm. Using these threshold values, extreme value calculations were performed to predict levels of acenaphthene that a certain percentage of the population could detect. These calculations are shown below:

Percent of Population Able to Detect Odor	Concentration of Acenaphthene (ppm)
20	2.6×10^{-2}
10	1.4×10^{-2}
1	1.9×10^{-3}
0.1	2.1×10^{-4}

Synergism and/or Antagonism

Two studies were conducted to investigate the effect of acenaphthene on the activity of dimethylnitrosamine demethylase (DMN-demethylase), the liver enzyme that demethylates DMN, a known carcinogen. Argus, et al. (1971) and Arcos,

et al. (1976) injected male weanling rats intraperitoneally with acenaphthene at a concentration equimolar to 40 mg of 20 methylcholanthrene/kg body weight. Twenty-four hours later, the animals were sacrificed and the liver microsomes assayed for DMN-demethylase activity. Acenaphthene showed a zero (Argus, et al. 1971) and a five percent (Arcos, et al. 1976) repression of the DMN demethylase levels over control rats with the same birth date. The difference in enzyme activity for the two studies may have been due to a modification of formaldehyde detection methods (Venkatesan, et al. 1968). In these studies, no value below ten percent was considered as significant activity. Arcos, et al. (1976) noted that demethylation is a requirement for carcinogenesis by DMN, and thus it is possible that acenaphthene may slightly inhibit DMN carcinogenesis.

Buu-Hoi and Hien-Do-Phouc (1969) investigated the effect of acenaphthene and other polycyclic aromatic hydrocarbons (PAHs) on the activity of zoxazolamine hydroxylase. Male Wistar rats were injected intraperitoneally with 20 mg/kg acenaphthene in corn oil, followed one week later by 90 mg/kg zoxazolamine. The mean paralysis time of treated rats was found to be significantly greater ($p < 0.01$) than that of vehicle-injected animals. The authors interpreted these results as an indication that acenaphthene retards the detoxification of zoxazolamine, which ordinarily proceeds via hydroxylation.

Teratogenicity

No information was found concerning the teratogenicity of acenaphthene.

Mutagenicity

The only data found on the mutagenicity of acenaphthene were four studies using microorganisms as the indicator system (Clark, 1953a,b; Gibson, et al. 1978; Guerin, et al. 1978). No mutagenicity was observed in any of the procedures used. Clark (1953a) studied the effect of acenaphthene on the recombination rate of two auxotrophic Bacterium coli (E. coli) strains. Acenaphthene was found to have no appreciable effect upon the recombination rate of either strain, as indicated by the low level of prototroph induction. Acenaphthene did induce pleomorphism, but not the filamentous "large" form which has been correlated with gene recombination. No metabolic activation was used in this study and the dose of acenaphthene administered was not specified. In a later study, Clark (1953b) tested acenaphthene for mutagenicity by exposing Micrococcus pyogenes var. aureus strain FDA209 to a saturated solution of acenaphthene in a water-based nutrient broth without a metabolic activation system. When induction of mutants resistant to penicillin or streptomycin was assessed, acenaphthene did not demonstrate any mutagenic effects.

Two mutagenicity studies performed using Salmonella typhimurium gave negative or inconclusive results. Guerin, et al. (1978) isolated an acenaphthene-containing aromatic subfraction from shale-derived crude oil and tested it for mutagenicity using S. typhimurium TA98. No revertants were observed with or without rat liver activation. Gibson, et al. (1978) exposed S. typhimurium strains to 200 to 2000

μg of acenaphthene dissolved in dimethylsulfoxide after first irradiating the acenaphthene samples with ^{60}Co to simulate (or replace) liver microsome activation. Unfortunately, the results were erratic with major toxicity observed at all dose levels tested. This toxicity obscured any assessment of mutagenicity.

The studies discussed above were the only ones found in the literature that examined the mutagenic potential of acenaphthene. A fifth study (Harvey and Halonen, 1968) examined the binding of acenaphthene to a variety of biologically important compounds as part of an unsuccessful attempt to correlate the nucleoside-binding activity of various chemicals with their carcinogenic potential. Acenaphthene showed significant binding constants for caffeine and riboflavin, but not for nucleosides.

Other Cellular Effects

The most thoroughly investigated effect of acenaphthene is its ability to produce nuclear and cytological changes in microbial and plant species. Most of these changes, such as an increase in cell size and DNA content, are associated with disruption of the spindle mechanism during mitosis and the resulting induction of polyploidy. While there is no known correlation between these effects and the biological impact of acenaphthene on mammalian cells, these effects are reported in this document because they are the only substantially investigated effects of acenaphthene.

Ten experiments examining the effect of acenaphthene on plants and eight others involving the effects upon micro-

organisms are discussed in the following sections. A summary of these data is presented in Table 1.

Plants: Kostoff (1938a) exposed Nicotiana longiflora shoots to vapors from acenaphthene crystals and examined the shoots for effects on mitosis and/or meiosis. The exposure induced tetraploid and octaploid shoots, which produced seeds of new polyploid plants. The polyploidizing effect of acenaphthene vapor increased with increases in the length of exposure or the number of particles used. Kostoff (1938b) also tested the effect of acenaphthene on the branches of floral buds of nine Nicotiana species. Meiosis in the buds proceeded abnormally also, with the bivalent chromosomes failing to arrange correctly on the equatorial plate. They tended to spread into the cytoplasm singly or in groups, resulting in a variable number of chromosomes per nuclei at the end of the second division. Fifty to one-hundred percent of the pollen produced by the end of meiosis was abortive.

In the same study, Kostoff (1938b) covered germinating seeds from a variety of plants with acenaphthene crystals to study the effects on mitosis. Cereals and grasses (wheat, rye, barley, oat, maize, and rice) showed slow growth and abnormal roots and leaf formations after four to eight days. Legumes evidenced these effects after 6 to 12 days, while compositae reacted in a time period midway between the other two groups. Mitosis in these seedlings proceeded abnormally; the spindle mechanism was inhibited and the chromosomes

TABLE 1
Summary of Polyploid and Other Mitotic Effects Induced by Acenaphthene in Plants and Microorganisms

ORGANISM	TREATMENT	EFFECTS NOTED	REFERENCE
Plants:			
Nicotiana shoots	Vapors	Stable polyploidy; abnormal, abortive meiosis	Kostoff, 1938a, b
Cereal, grass legume, and compositae seeds	Crystals (4-12 days)	Abnormal mitosis, spindle mechanism inhibited	Kostoff, 1938b
Cherry-mazzard hybrid seeds	Powder (10 hours)	Seed germination and growth inhibited; no polyploidy	Zhukov, 1971
Allium cepa L.	Saturated solution (2-5 days)	Chromosome fragmentation, polyploidy	D'Amato, 1949
Allium cepa L., A. sativum	Treatment unspecified	Frequency of division retarded, multiple prophase	Mookerjee, 1973
Allium fistulosum, Colchicum roots	Crystals wrapped in moist filter paper (4-20 days)	C-mitosis, polyploidy, root-tip swellings	Levan, 1940
Allium root cells	Vapors (12-96 hours)	Random cell wall development	Mesquita, 1967

TABLE 1 (cont.)

ORGANISM	TREATMENT	EFFECTS NOTED	REFERENCE
Binucleate pollen	Vapors	Spindle inhibited, division stopped at metaphase	Dyer, 1966
Tradescantia pollen	Vapors	Spindle disturbed	Swanson, 1940
Tradescantia stamen hairs	Saturated solution (2-4.5 hours)	No polyploidy, no chromosomes in metaphase	Nebel, 1938
Fungi: Basidomycetes	Vapors	Mitotic frequency decrease; growth, pigment formation, differentiation and morphology changes	Hoover, 1972
Basidiobolus ranarum hyphae	Vapors (6-18 hours)	Alterations in nuclear division	Hoover and Liberta, 1974
Pythium aphanidermatum hyphae	Vapors or supersaturated solution (12 hours)	Nuclear division arrested; pyknosis	Seshadri and Payak, 1970
Yeast	10^{-1} to 3×10^{-7} mol Solution	No lethality or c-mitosis	Levan and Sandwall, 1943
Candida scottii	0.2-1.0% agar	Increase in cell size, nucleus, DNA content	Imshenetsky, et al. 1966

TABLE 1 (cont.)

ORGANISM	TREATMENT	EFFECTS NOTED	REFERENCE
Bacteria:			
Mycobacterium rubrum	Vapors from 10-20 mg crystals	Elongation and thickening of cells; unstable polyploidy	Imshenetsky and Zhil'tsova, 1973
Rhizobium	Vapors	Increase in DNA content; change in biochemical properties	Avvakumova, et al. 1975
Algae:			
Chara globularis; *Nitella flagelliformis*	Saturated solution (12-120 hours)	Number of cells in mitosis reduced; chromosomes clumped at metaphase; chromosomes doubled	Sarma and Tripathi, 1976a, b

were not arranged on the equitorial plate. Failure of the chromosomes to move to the poles resulted in polyploidy.

Zhukov (1971) investigated the effect of acenaphthene on plant seeds. He treated "cherry-mazzard hybrid" seeds with acenaphthene powder for ten hours. Seed germination and seedling growth were inhibited, but no polyploidal cells were found in the plant roots.

Four investigators performed experiments with acenaphthene and Allium plants. When treated with saturated solutions of acenaphthene in either tap or distilled water for two to five days, Allium cepa L. demonstrated intense chromosome fragmentation (D'Amato, 1949). Fragmenting effects on diploid and polyploidized nuclei in the resting stage were noted, as were centromere effects on the metaphase chromosomes and, occasionally, on chromatids at anaphase. In a later study (Mookerjee, 1973), acenaphthene exposure (concentration unspecified) was found to retard the frequency of division of Allium cepa and Allium sativum. Multiple prophase was observed in A. cepa.

Levan (1940) dusted Allium fistulosum and Colchicum roots with acenaphthene crystals and then wrapped the plants in moist filter paper. After four days of growth, the spindles were altered and the centromeres inactivated: this process has been termed "c-mitosis" because a similar effect occurs with colchicine treatment. Tetraploid and octaploid cells were formed within 14 to 20 days, resulting in the formation of root-tip swellings (c-tumors) in Allium. Mesquita (1967) also investigated the effects of acenaphthene on Allium

root cells. He exposed A. cepa root tips to acenaphthene vapor at room temperature for 12 to 96 hours. The reassembling of the phragmoplast elements (small pieces of the endoplasmic reticulum and Golgi bodies) in the equitorial region was inhibited, but the fusion of these elements in other parts of the cell was unimpaired. The result was the random development of cell walls.

To investigate the effect of acenaphthene on mitosis, Dyer (1966) exposed plant species with binucleate pollen (such as Bellevalia romana, Tulbaghia natalensis, and Antirrhinum majus) to vapor from acenaphthene crystals. He found that all cells remained at metaphase, with anaphase being inhibited due to an inhibition of the mitotic spindle. Swanson (1940) also observed effects on mitosis in plant pollen. He scattered acenaphthene crystals on the bottom of a petri dish in which Tradescantia pollen was incubated. The vapors acted by disturbing the spindle mechanism so that the chromosomes remained in place after division. Nebel (1938) examined the effect of acenaphthene on mitosis in plant hairs by treating stamen hairs of Tradescantia with a saturated solution of acenaphthene in liquid media for 2 and 4.5 hours. He found no polyploid cells and no nuclei showing chromosomes in a metaphase condition.

Microorganisms: Several experiments have been performed to investigate the effect of acenaphthene on microorganisms. Hoover (1972) exposed 37 species of Basidiomycetes to acenaphthene vapors or media containing acenaphthene at unspecified dose levels in order to examine effects on growth, pigment,

morphology, nuclear division, and fruit body formation. As the treatment time increased, changes in nuclear division became more pronounced, with a concurrent decrease in the mitotic frequency. Growth, pigment formation, differentiation, and colonial and cellular morphology were affected by acenaphthene treatment. A delay or prevention of light-induced fruitbody formation occurred in one species; two species developed greatly enlarged fruitbodies as a result of this treatment. The genetic stability of these phenotypic changes was not demonstrated, however.

In a later experiment, Hoover and Liberta (1974) exposed hyphae cultures of the fungus Basidiobolus ranarum to acenaphthene vapors for 6 to 18 hours. At the end of 18 hours, gross alterations in nuclear division were observed and the spindle fibers were rendered unstainable. The time required for division was significantly increased in acenaphthene-treated cells. The effect of acenaphthene on fungi was also investigated by Seshadri and Payak (1970). They exposed hyphae of Pythium aphanidermatum to acenaphthene vapors or to a supersaturated solution of acenaphthene for 12 hours. The vapors proved "instrumental" in arresting the progress of nuclear division. A marked increase in the size and sporangia nuclei was noted, and the nuclei showed various degrees of pyknosis and shape irregularity.

Levan and Sandwall (1943) examined the effect of varying concentrations of acenaphthene (10^{-1} to $3x10^{-7}$ mol solution in ethanol) on wort yeast cell cultures. Even at the highest concentration, there was no lethality or effect

on cell propagation. The authors concluded that the c-mitotic action demonstrated by acenaphthene in higher plants was not observable in yeast. Polyploidy was induced, however, in the yeast Candida scottii (a yeast without a sexual cycle) when treated with 0.2 percent and 1.0 percent acenaphthene added to agar medium (Imshenetsky, et al. 1966). The size of the cell and the nucleus were both increased in the treated cultures, and there was also a higher dry biomass for these cells. The DNA content (µg per cell) was higher in acenaphthene-treated cells, although the difference between experimental and control cultures decreased as the cultures aged.

Imshenetsky and Zhil'tsova (1973) attempted to produce "polyploid-like" cells by exposing Mycobacterium rubrum to vapors from 10 to 20 mg acenaphthene. When the vapors were used alone for treatment, there was no increase in the size of the cells, nor any indication of the induction of polyploidy. When the cells were treated with water or EDTA to increase membrane permeability, acenaphthene vapor treatment caused elongation and thickening of cells, with a longer development cycle; these "polyploid-like" changes were found to be unstable, however. In another experiment with bacteria, Avvakumova, et al. (1975) treated Rhizobium (nodule-forming pea bacteria) with acenaphthene vapors (dose unspecified) to induce polyploidy. The authors found an acenaphthene-associated increase in cellular DNA content and biomass, as well as a change in biochemical properties, e.g., the ability to assimilate carbohydrates and/or organic acids.

Acenaphthene has also been shown to affect mitosis in two species of algae. Sarma and Tripathi (1976a,b) treated Chara globularis and Nitella flagelliformis with a saturated solution of acenaphthene for 12 to 120 hours. The number of cells in mitosis was reduced by 40 percent, and the chromosomes were seen to clump at metaphase after 120 hours. Nine percent of the C. globularis cells showed complete chromosome doubling by the end of the treatment period.

Carcinogenicity

Very little work has been done to determine whether acenaphthene may have carcinogenic properties. Neukomm (1974) reported negative results in a predictive test for carcinogenicity based upon neoplastic induction in the newt Triturus cristatus. Ten animals were injected subcutaneously with acenaphthene (dose and solvent not reported) in the fleshy part of the tail along the vertebral axis. Samples of the injection site were removed at 7 and 14 days, and the tissues were examined for neoplastic infiltration in the epidermis and the development or regression of diffuse tumors. Neoplastic lesions were divided into three categories depending on the size of the lesion and assigned a numerical coefficient accordingly: large (1.0), intermediate (0.5), and limited (0.25). Calculation of a neoplastic index by summing the coefficients of all lesions and dividing by the number of observed animals gave an index for acenaphthene of 0.0, indicating a lack of neoplastic induction in the newt.

Neukomm (1974) discussed the reliability of this test by drawing a correlation between positive index values for a few polycyclic aromatic hydrocarbons and the carcinogenicity of these same compounds for mouse skin. These limited comparisons, however, are not sufficient to establish the value of this test for predicting carcinogenicity in mammalian systems.

The only other carcinogenicity studies in the literature involving acenaphthene considered it as one component of a complex mixture of PAHs. It is impossible in these studies to sort out the relative contribution of acenaphthene versus other hydrocarbons in the mixture, so no real conclusions can be drawn. Akin, et al. (1976) isolated some polycyclic hydrocarbon-rich fractions of the neutral portion of cigarette smoke condensate (CSC) and tested them for tumor promotion on female mouse skin, using 7,12-dimethylbenz(a)anthracene (DMBA) as the initiator. Animals were painted once with 125 µg DMBA on dorsal skin; three to four weeks later the fractions were applied five times a week for 13 months. The fraction containing acenaphthene, pyrene, phenanthrene, and other PAHs, showed no significant tumor-promoting activity over controls treated with DMBA and acetone. This result was surprising in view of the fact that Scribner (1973) had demonstrated the tumor-promoting ability of pyrene and phenanthrene.

In 1962, Hoffman and Wynder found that benzene extracts of gasoline exhaust condensates were carcinogenic in mouse skin painting tests. This study is of interest considering

a later study by Grimmer, et al. (1977) which showed that acenaphthene was present in an unspecified concentration in the benzene extracts of gasoline exhaust condensate. Unfortunately, the possible contribution of acenaphthene to the observed carcinogenicity (Hoffman and Wynder, 1962) cannot be determined from this limited evidence.

CRITERION FORMULATION

Existing Guidelines and Standards

No existing guidelines or standards were found.

Current Levels of Exposure

Virtually no information is available concerning the prevalence or concentration of acenaphthene in the environment. Acenaphthene has been detected in cigarette smoke (Harke, et al. 1976; Severson, et al. 1976), automobile exhaust (Grimmer, et al. 1977), and in urban air (Cleary, 1962) and is present in coal tar and several fossil fuel oils. It has also been reported in wastewater from petrochemical, pesticide, and wood presevative industries (U.S. EPA, 1978b) and detected in water from a river in the Netherlands (Meijers and Van der Leer, 1976).

Special Groups at Risk

Individuals working with coal tar and/or its products face a possible risk due to increased exposure to acenaphthene, although no data are available to estimate this risk.

Basis and Derivation of Criterion

So little research has been performed on acenaphthene that its mammalian and human health effects are virtually unknown. The two toxicity studies available (Knobloch, et al. 1969; Reshetyuk, et al. 1970) are inadequate for use as the basis of a criterion due to deficiencies in the experimental designs (lack of controls, small number of animals, etc.). Therefore, until more toxicological data are generated, particularly teratogenic data in view of the effects of acenaphthene on cell division, an interim

criterion based upon organoleptic data is proposed. The lowest levels eliciting human responses were reported at 0.022 to 0.22 ppm (Lillard and Powers, 1975), and thus 0.02 ppm (0.02 mg/l) is the recommended criterion.

Since the recommended criterion is based on organoleptic effects and is not a toxicological assessment, the consumption of fish and shellfish products will not be considered as a route of exposure.

It must be emphasized, however, that this value is not related to health effects and that the significance of odor thresholds is unknown. This criterion will need to be reviewed once more toxicological data are available.

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